What Is Claimed Is:

1. An ApoA-I agonist comprising:

(i) a 15 to 29-residue peptide or peptide analogue which forms an amphipathic α-helix in the presence of lipids and which comprises the structural formula (I):

$$Z_1\text{-}X_1\text{-}X_2\text{-}X_3\text{-}X_4\text{-}X_5\text{-}X_6\text{-}X_7\text{-}X_8\text{-}X_9\text{-}X_{10}\text{-}X_{11}\text{-}X_{12}\text{-}X_{13}\text{-}X_{14}\text{-}X_{15}\text{-}X_{16}\text{-}X_{17}\text{-}X_{18}\text{-}X_{19}\text{-}X_{20}\text{-}X_{21}\text{-}X_{22}\text{-}X_{23}\text{-}Z_2$$

or a pharmaceutically acceptable salt thereof, wherein:

X_1 is Pro (P), Ala (A), Gly (G), Gln (Q), Asn (N), Asp (D) or D-Pro (p);

X_2 is an aliphatic residue;

X_3 is Leu (L) or Phe (F);

X_4 is an acidic residue;

X_5 is Leu (L) or Phe (F);

X_6 is Leu (L) or Phe (F);

X_7 is a hydrophilic residue;

X_8 is an acidic or a basic residue;

X_9 is Leu (L) or Gly (G);

X_{10} is Leu (L), Trp (W) or Gly (G);

X_{11} is a hydrophilic residue;

X_{12} is a hydrophilic residue;

X_{13} is Gly (G) or an aliphatic residue;

X_{14} is Leu (L), Trp (W), Gly (G) or Nal;

X_{15} is a hydrophilic residue;

X_{16} is a hydrophobic residue;

X_{17} is a hydrophobic residue;

X_{18} is Gln (Q), Asn (N) or a basic residue;

X_{19} is Gln (Q), Asn (N) or a basic residue;

X_{20} is a basic residue;

X_{21} is an aliphatic residue;

X_{22} is a basic residue;

X_{23} is absent or a basic residue;

Z_1 is H_2N- or RC(O)NH-;

Z_2 is -C(O)NRR, -C(O)OR or -C(O)OH or a salt thereof;

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl, (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl, 6-26 membered alkheteroaryl or a 1 to 7-residue peptide or peptide analogue;

each " - " between residues X_n independently designates an amide linkage, a substituted amide linkage, an isostere of an amide or an amide mimetic; or

(ii) a deleted from of structural formula (I) in which at least one and up to eight of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17}, X_{18}, X_{19}, X_{20}, X_{21} and X_{22} are deleted; or

(iii) an altered form of structural formula (I) in which at least one of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17}, X_{18}, X_{19}, X_{20}, X_{21}, X_{22} or X_{23} is conservatively substituted with another residue.

2. The ApoA-I agonist of Claim 1 which exhibits at least about 38% LCAT-activation activity as compared with human ApoA-I.

3. The ApoA-I agonist of Claim 1 which is the altered form of structural formula (I).

4. The ApoA-I agonist of Claim 3 in which the hydrophobic residues are fixed according to structural formula (I) and at least one non-fixed residue is conservatively substituted with another residue.

5. The ApoA-I agonist of Claim 4 in which:
X_1 is Pro (P), D-Pro (p), Gly (G) or Ala (A);
X_2 is Ala (A), Leu (L) or Val (V);
X_3 is Leu (L) or Phe (F);

X_5 is Leu (L) or Phe (F);
X_6 is Leu (L) or Phe (F);
X_9 is Leu (L) or Gly (G);
X_{10} is Leu (L), Trp (W) or Gly (G);
X_{13} is Leu (L), Gly (G) or Aib;
X_{14} is Leu, Nal, Trp (W) or Gly (G);
X_{16} is Ala (A), Nal, Trp (W), Gly (G), Leu (L) or Phe (F);
X_{17} is Leu (L), Gly (G) or Nal;
X_{21} is Leu (L); and

at least one of X_4, X_7, X_8, X_{11}, X_{12}, X_{15}, X_{18}, X_{19}, X_{20}, X_{22} and X_{23} is conservatively substituted with another residue.

6. The ApoA-I agonist of Claim 3 in which the hydrophilic residues are fixed according to structural formula (I) and at least one non-fixed residue is conservatively substituted with another residue.

7. The ApoA-I agonist of Claim 6 in which:
X_4 is Asp (D) or Glu (E);
X_7 is Lys (K), Arg (R) or Orn;
X_8 is Asp (D) or Glu (E);
X_{11} is Asn (N) or Gln (Q);
X_{12} is Glu (E) or Asp (D);
X_{15} is Asp (D) or Glu (E);
X_{18} is Gln (Q), Asn (N), Lys (K) or Orn;
X_{19} is Gln (Q), Asn (N), Lys (K) or Orn;
X_{20} is Lys (K) or Orn;
X_{22} is Lys (K) or Orn;
X_{23} is absent or Lys (K); and

at least one of X_1, X_2, X_3, X_5, X_6, X_9, X_{10}, X_{13}, X_{14}, X_{16}, X_{17} and X_{21} is conservatively substituted with another residue.

8. The ApoA-I agonist of Claim 7 in which X_3 is Leu (L) or Phe (F), X_6 is Phe (F), X_9 is Leu (L) or Gly (G), X_{10} is Leu (L) or Trp (W) or Gly (G) and at least one of X_1, X_2, X_5, X_{13},

X_{14}, X_{16}, X_{17} and X_{21} is conservatively substituted with another residue.

9. The ApoA-I agonist of Claim 5 or 7 in which the substituting residue is classified within the same sub-category as the substituted residue.

10. The ApoA-I agonist of Claim 1 which is the deleted form of structural formula (I).

11. The ApoA-I agonist of Claim 10 in which one helical turn of the peptide or peptide analogue is deleted.

12. The ApoA-I agonist of Claim 1 which is a 22-23 residue peptide or peptide analogue of structural formula (I).

13. The ApoA-I agonist of Claim 12 in which:
the "-" between residues designates -C(O)NH-;
Z_1 is H_2N-; and
Z_2 is -C(O)OH or a salt thereof.

14. The ApoA-I agonist of Claim 13, in which:
X_1 is Pro (P), Ala (A), Gly (G), Asn (N), Gln (Q), Asp (D) or D-Pro (p);
X_2 is Ala (A), Val (V) or Leu (L);
X_3 is Leu (L) or Phe (F);
X_4 is Asp (D) or Glu (E);
X_5 is Leu (L) or Phe (F);
X_6 is Leu (L) or Phe (F);
X_7 is Lys (K), Arg (R) or Orn;
X_8 is Asp (D) or Glu (E);
X_9 is Leu (L) or Gly (G);
X_{10} is Leu (L), Trp (W) or Gly (G);
X_{11} is Asn (N) or Gln (Q);
X_{12} is Glu (E) or Asp (D);

X_{13} is Gly (G), Leu (L) or Aib;
X_{14} is Leu (L), Nal, Trp (W) or Gly (G);
X_{15} is Asp (D) or Glu (E);
X_{16} is Ala (A), Nal, Trp (W), Leu (L), Phe (F) or Gly (G);
X_{17} is Gly (G), Leu (L) or Nal;
X_{18} is Gln (Q), Asn (N), Lys (K) or Orn;
X_{19} is Gln (Q), Asn (N), Lys (K) or Orn;
X_{20} is Lys (K) or Orn;
X_{21} is Leu (L);
X_{22} is Lys (K) or Orn; and
X_{23} is absent or Lys (K).

15. The ApoA-I agonist of Claim 14, in which X_{23} is absent.

16. The ApoA-I agonist of Claim 13 or 14, in which one of X_{18} or X_{19} is Gln (Q) or Asn (N) and the other of X_{18} or X_{19} is Lys (K) or Orn.

17. The ApoA-I agonist of Claim 14 in which each of X_9, X_{10}, X_{13}, X_{14}, X_{15} and X_{17} is other than Gly (G).

18. The ApoA-I agonist of Claim 14 in which one of X_9, X_{10}, X_{13}, X_{14}, X_{15} and X_{17} is Gly (G) and the others are other than Gly (G).

19. The ApoA-I agonist of Claim 1 which is selected from the group consisting of:

peptide 1	PVLDLFRELLNELLEZLKQKLK	(SEQ ID NO:1)
peptide 2	GVLDLFRELLNELLEALKQKLKK	(SEQ ID NO:2)
peptide 3	PVLDLFRELLNELLEWLKQKLK	(SEQ ID NO:3)
peptide 4	PVLDLFRELLNELLEALKQKLK	(SEQ ID NO:4)
peptide 5	pVLDLFRELLNELLEALKQKLKK	(SEQ ID NO:5)
peptide 6	PVLDLFRELLNEXLEALKQKLK	(SEQ ID NO:6)

peptide 7	PVLDLFKELLNELLEALKQKLK	(SEQ ID NO:7)
peptide 8	PVLDLFRELLNEGLEALKQKLK	(SEQ ID NO:8)
peptide 9	PVLDLFRELGNELLEALKQKLK	(SEQ ID NO:9)
peptide 10	PVLDLFRELLNELLEAZKQKLK	(SEQ ID NO:10)
peptide 11	PVLDLFKELLQELLEALKQKLK	(SEQ ID NO:11)
peptide 12	PVLDLFRELLNELLEAGKQKLK	(SEQ ID NO:12)
peptide 13	GVLDLFRELLNEGLEALKQKLK	(SEQ ID NO:13)
peptide 14	PVLDLFRELLNELLEALOQOLO	(SEQ ID NO:14)
peptide 15	PVLDLFRELWNELLEALKQKLK	(SEQ ID NO:15)
peptide 16	PVLDLLRELLNELLEALKQKLK	(SEQ ID NO:16)
peptide 17	PVLELFKELLQELLEALKQKLK	(SEQ ID NO:17)
peptide 18	GVLDLFRELLNELLEALKQKLK	(SEQ ID NO:18)
peptide 19	pVLDLFRELLNEGLEALKQKLK	(SEQ ID NO:19)
peptide 20	PVLDLFREGLNELLEALKQKLK	(SEQ ID NO:20)
peptide 21	pVLDLFRELLNELLEALKQKLK	(SEQ ID NO:21)
peptide 22	PVLDLFRELLNELLEGLKQKLK	(SEQ ID NO:22)
peptide 23	PLLELFKELLQELLEALKQKLK	(SEQ ID NO:23)
peptide 24	PVLDLFRELLNELLEALQKKLK	(SEQ ID NO:24)
peptide 25	PVLDFFRELLNEXLEALKQKLK	(SEQ ID NO:25)
peptide 26	PVLDLFRELLNELLELLKQKLK	(SEQ ID NO:26)
peptide 27	PVLDLFRELLNELZEALKQKLK	(SEQ ID NO:27)
peptide 28	PVLDLFRELLNELWEALKQKLK	(SEQ ID NO:28)
peptide 29	AVLDLFRELLNELLEALKQKLK	(SEQ ID NO:29)
peptide 123	QVLDLFRELLNELLEALKQKLK	(SEQ ID NO:123)
peptide 124	PVLDLFOELLNELLEALOQOLO	(SEQ ID NO:124)
peptide 125	NVLDLFRELLNELLEALKQKLK	(SEQ ID NO:125)
peptide 126	PVLDLFRELLNELGEALKQKLK	(SEQ ID NO:126)
peptide 127	PVLDLFRELLNELLELLKQKLK	(SEQ ID NO:127)
peptide 128	PVLDLFRELLNELLEFLKQKLK	(SEQ ID NO:128)
peptide 129	PVLELFNDLLRELLEALQKKLK	(SEQ ID NO:129)
peptide 130	PVLELFNDLLRELLEALKQKLK	(SEQ ID NO:130)
peptide 131	PVLELFKELLNELLDALRQKLK	(SEQ ID NO:131)
peptide 132	PVLDLFRELLENLLEALQKKLK	(SEQ ID NO:132)

peptide **133**	PVLELFERLLEDLLQALNKKLK	(SEQ ID NO:133)
peptide **134**	PVLELFERLLEDLLKALNQKLK	(SEQ ID NO:134)
peptide **135**	DVLDLFRELLNELLEALKQKLK	(SEQ ID NO:135)
peptide **136**	PALELFKDLLQELLEALKQKLK	(SEQ ID NO:136)
peptide **137**	PVLDLFRELLNEGLEAZKQKLK	(SEQ ID NO:137)
peptide **138**	PVLDLFRELLNEGLEWLKQKLK	(SEQ ID NO:138)
peptide **139**	PVLDLFRELWNEGLEALKQKLK	(SEQ ID NO:139)
peptide **140**	PVLDLFRELLNEGLEALOQOLO	(SEQ ID NO:140)
peptide **141**	PVLDFFRELLNEGLEALKQKLK	(SEQ ID NO:141)
peptide **142**	PVLELFRELLNEGLEALKQKLK	(SEQ ID NO:142)

and the N-terminal acylated and/or C-terminal amidated or esterified forms thereof, wherein X is Aib; Z is Nal; and O is Orn.

20. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (II):

(II) $HH\{LL_m\text{-}HH\}_nLL_m\text{-}HH$

or a pharmaceutically acceptable salt thereof, wherein:

each m is independently an integer from 0 to 1;

n is an integer from 0 to 10;

each "HH" is independently a peptide or peptide analogue according to Claim 1;

each "LL" is independently a bifunctional linker; and

each " - " independently designates a covalent linkage.

21. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (III):

(III) $X\text{-}N_{ya}\text{-}X_{(ya-1)}\text{-}(N_{yb}\text{-}X_{(yb-1)})_p$

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently HH-(LL$_m$-HH)$_n$-LL$_m$-HH;

each HH is independently a core peptide of structure (I) or an analogue or mutated, truncated, internally deleted or extended form thereof as described herein;

each LL is independently a bifunctional linker;

each m is independently an integer from 0 to 1;

each n is independently an integer from 0 to 8;

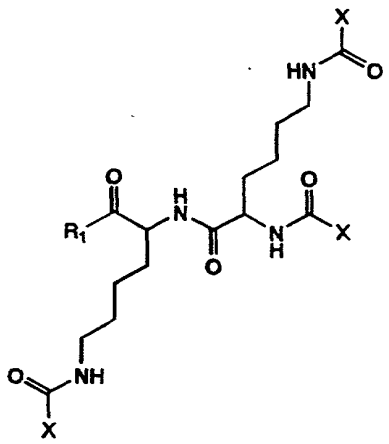
N_{ya} and N_{yb} are each independently a multifunctional linking moiety where y_a and y_b represent the number of functional groups on N_{ya} and N_{yb}, respectively;

each y_a or y_b is independently an integer from 3 to 8;

p is an integer from 0 to 7; and

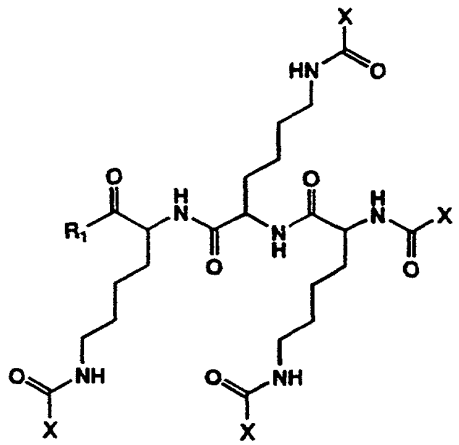
each "–" independently designates a covalent bond.

22. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (IV) or (V):



(IV)

-OR-



(V)

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently HH-(LL$_m$-HH)$_n$LL$_m$-HH;

each HH is independently a peptide or peptide analogue according to Claim 1;

each LL is independently a bifunctional linker;

each n is independently an integer from 0 to 1;

each m is independently an integer from 0 to 8;

R_1 is -OR or -NRR; and

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl; (C_5-C_{20}) aryl (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl.

23. The multimeric ApoA-I agonist of Claim 20, 21 or 22 in which the bifunctional linker is cleavable.

24. The ApoA-I multimeric agonist of Claim 20, 21 or 22 in which n is 0.

25. The multimeric ApoA-I agonist of Claim 24 in which m is 0.

26. The multimeric ApoA-I agonist of Claim 20, 21 or 22 in which each HH is independently a peptide according to Claim 13.

27. The multimeric ApoA-I agonist of Claim 20, 21 or 22 in which each HH is independently a peptide according to Claim 14.

28. The multimeric ApoA-I agonist of Claim 20, 21 or 22 in which each HH is independently a peptide according to Claim 19.

29. An ApoA-I agonist-lipid complex comprising an ApoA-I agonist and a lipid, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist according to Claim 20, a multimeric

ApoA-I agonist according to Claim 21, or a multimeric ApoA-I agonist according to Claim 22.

30. The ApoA-I agonist-lipid complex of Claim 29 in which the ApoA-I agonist is a peptide according to Claim 12.

31. The ApoA-I agonist-lipid complex of Claim 29 in which the ApoA-I agonist is a peptide according to Claim 13.

32. The ApoA-I agonist-lipid complex of Claim 29 in which the ApoA-I agonist is a peptide according to Claim 14.

33. The ApoA-I agonist-lipid complex of Claim 29 in which the ApoA-I agonist is a peptide according to Claim 19.

34. The ApoA-I agonist-lipid complex of Claim 29 in which the lipid is sphingomyelin.

35. The ApoA-I agonist-lipid complex of Claim 29 which is in the form of a lyophilized powder.

36. The ApoA-I agonist-lipid complex of Claim 29 which is in the form of a solution.

37. A pharmaceutical composition comprising an ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist according to Claim 20, a multimeric ApoA-I agonist according to Claim 21, or a multimeric ApoA-I agonist according to Claim 22.

38. The pharmaceutical composition of Claim 37 in which the ApoA-I agonist is a peptide according to Claim 12.

39. The pharmaceutical composition of Claim 37 in which the ApoA-I agonist is a peptide according to Claim 13.

40. The pharmaceutical composition of Claim 37 in which the ApoA-I agonist is a peptide according to Claim 14.

41. The pharmaceutical composition of Claim 37 in which the ApoA-I agonist is a peptide according to Claim 19.

42. The pharmaceutical composition of Claim 37, 38, 39, 40 or 41, in which the ApoA-I agonist is in the form of an ApoA-I agonist-lipid complex, said complex comprising the ApoA-I agonist and a lipid.

43. The pharmaceutical composition of Claim 42 in which the ApoA-I agonist-lipid complex is in the form of a lyophilized powder.

44. A method of treating a subject suffering from a disorder associated with dyslipidemia, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

45. The method of Claim 44 in which the ApoA-I agonist is in the form of a pharmaceutical composition, said composition comprising the ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent.

46. The method of Claim 44 in which the ApoA-I agonist is in the form of an ApoA-I agonist-lipid complex, said complex comprising the ApoA-I agonist and a lipid.

47. The method of Claim 44 in which the disorder associated with dyslipidemia is hypercholesterolemia.

48. The method of Claim 44 in which the disorder associated with dyslipidemia is cardiovascular disease.

49. The method of Claim 44 in which the disorder associated with dyslipidemia is atherosclerosis.

50. The method of Claim 44 in which the disorder associated with dyslipidemia is restenosis.

51. The method of Claim 44, in which the disorder associated with dyslipidemia is HDL or ApoA-I deficiency.

52. The method of Claim 44, in which the disorder associated with dyslipidemia is hypertriglyceridemia.

53. The method of Claim 44, in which the disorder associated with dyslipidemia is metabolic syndrome.

54. A method of treating a subject suffering from septic shock, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

55. The method of Claim 44 or 54 in which said subject is a human.

56. The method of Claim 44 or 54 in which about 0.5 mg/kg to about 100 mg/kg ApoA-I agonist is administered to said subject.